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Inpatient Management of Patients With Cirrhosis

Presentation and Evaluation

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Cirrhosis affects millions of U.S. adults and costs the U.S. health care system upward of $6 billion annually. Cirrhosis is underrecognized, and the only cure is transplantation. Complications, including bleeding, infection, ascites, and renal injury, contribute to high rates of hospitalization, readmission, and mortality in this population. Evidence-based practices and guidelines offer quality recommendations for clinicians, but many of these guidelines have changed recently. This article provides an update on the current guidelines for the inpatient management of cirrhosis.

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Physician Writer
Shari Rogal, MD, MPH
Center for Healthcare Evaluation, Research, and Promotion, VA Pittsburgh Healthcare System, and Departments of Medicine and Surgery, University of Pittsburgh, Pittsburgh, Pennsylvania

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CME Objective: To review current evidence on presentation, evaluation, diagnosis, management, and practice improvement for cirrhosis among inpatients.

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Liver disease affects 4.5 million people in the United States, with the most frequent causes being metabolic dysfunction–associated steatotic liver disease, alcohol, and viral hepatitis (1, 2). Cirrhosis (advanced liver disease) is the tenth leading cause of death in the United States, where management costs $6 billion annually (3). Despite the high costs and consequences of cirrhosis, less than half of patients with cirrhosis are diagnosed, leading to preventable morbidity and mortality (4, 5). Urgent, appropriate recognition and intervention can be life-saving. Thus, internal medicine physicians, particularly hospitalists, need to understand inpatient management of cirrhosis complications. The goals of inpatient cirrhosis management may include stabilization, risk stratification, management of complications, transition to the outpatient setting, transplant, or palliative care as well as education to prevent rehospitalization. This can be challenging due to the evolving nature of evidence and guidelines. This article summarizes key recommendations based on guidance published by the American Association for the Study of Liver Diseases (AASLD), the American College of Gastroenterology (ACG), the American Gastroenterological Association (AGA), the European Association for the Study of the Liver (EASL), and the American Society of Transplantation (AST) and offers key studies for consideration.

Presentation and Evaluation

How is the presence of cirrhosis assessed in inpatients?

Cirrhosis is defined histologically as irreversible, nodular fibrosis. Although biopsy has historically been the diagnostic gold standard, the advent and increasing popularity of noninvasive measures of fibrosis has obviated the need for biopsy in most cases. AASLD revised its guidelines to focus on noninvasive means of diagnosis and their implications for downstream management. Rather than using a binary definition of cirrhosis based on the presence of fibrotic nodules on biopsy specimens, AASLD offers guidance around continuous measures of liver stiffness (6, 7). The Baveno VII consensus conference recommended using the term "compensated advanced chronic liver disease" (cACLD) rather than the histologic term "cirrhosis" to recognize this shift away from histologic diagnosis. AASLD defined the likelihood of cACLD based on a combination of noninvasive measures of liver stiffness (elastography) and platelets. The literature and recommendations vary in the precise thresholds, with the Baveno VII group offering thresholds of less than 8 kPa to exclude cACLD and above 12 kPa to define it (8, 9) and AASLD recommending the cutoffs shown in **Figure 1** (10).

How is cirrhosis severity assessed in inpatients?

Once cirrhosis is diagnosed, it is critical to risk-stratify patients. To do so, AASLD guidance recommends using terms that reflect the natural history of cirrhosis, with "compensated cirrhosis" defined by a lack of clinically overt complications and "decompensated cirrhosis" defined by symptomatic disease that includes ascites, variceal bleeding, and encephalopathy (10). This delineation was based on studies showing that the 12-year median survival in compensated cirrhosis decreases to less than 2 years after decompensation (10). **Figure 2** illustrates how compensated and decompensated disease management differ; the former focuses on prevention of decompensation by removing risk factors and considering nonselective β-blockers (NSBBs), whereas the latter focuses on management of the specific events that lead to death (11).

AASLD guidelines specify how noninvasive measures, including laboratory-based testing and liver stiffness

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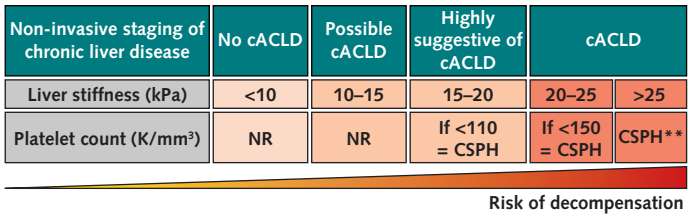
Figure 1. **AASLD recommendations for defining cACLD by noninvasive testing.**



AASLD = American Association for the Study of Liver Diseases; cACLD = compensated advanced chronic liver disease; CSPH = clinically significant portal hypertension. (Reproduced from Kaplan DE, Ripoll C, Thiele M, et al, AASLD practice guidance on risk stratification and management of portal hypertension and varices in cirrhosis, *Hepatology*, volume 79, issue 5, pages 1180–1211 [http://doi.org/10.1097/HEP.0000000000000647], with permission.)

assessment, can further risk-stratify patients with cirrhosis based on the presence or absence of clinically significant portal hypertension (CSPH) (6, 7). This is important because CSPH corresponds with risk for adverse outcomes (**Figure 1**). Approaches may include simple laboratory-based tests (such as the aspartate aminotransferase to platelet ratio index or the Fibrosis-4 Index) and/or elastography (such as FibroScan testing or magnetic resonance elastography) (6). CSPH can be diagnosed by direct measurement, with portal hypertension defined as a hepatic venous pressure gradient (HVPG) of 10 mm Hg or greater, radiographic signs (for example, reversal of portal flow on Doppler ultrasonography or collateral vessels), complications of liver disease (such as ascites, hepatic encephalopathy [HE], hepatopulmonary syndrome, hepatorenal syndrome [HRS], or variceal bleeding), or meeting a combination of elastography and platelet criteria (liver stiffness ≥25 kPa, or ≥20 kPa with platelet count $<150 \times 10^9$ cells/L) (8).

Several calculated scores are used for prognosticating and risk-stratifying patients with cirrhosis. The most frequently used is the Model for End-Stage Liver Disease (MELD) 3.0, which built on the existing MELD-Na score by adding extra points for female sex, albumin, and an interaction term between creatinine and albumin to bilirubin, sodium, creatinine, and international normalized ratio (INR), which were already included (12). MELD 3.0 has improved predictive capabilities for mortality and accounts for death on the waitlist and disparities for women (12). A score of 6 denotes 90-day survival of 98%, while a score above 40 indicates 90-day survival below 30%. A threshold of 15 has been used to define the point at which the benefits of transplant outweigh the risks (**Table 1**) (12, 13). MELD 3.0 was developed to predict short-term, population-level mortality for people with cirrhosis (12).

Although an initial work-up should include calculating the MELD 3.0 score in patients with known cirrhosis, it is important to note that MELD 3.0 was based on population-level modeling and may not apply to individual patients, who may have MELD scores that do not adequately reflect their degree of impairment and liver disease. Therefore, when patients have decompensated cirrhosis or hepatocellular carcinoma, a transplant hepatology team should generally be involved, regardless of the calculated MELD score.

AASLD recommends that evaluation for liver transplant be considered once a patient with cirrhosis has an index complication, such as ascites, HE, or variceal hemorrhage, or hepatocellular dysfunction results in a MELD score of 15 or higher (1-A) (13). This is based on a study that evaluated 12 996 adults listed for liver transplant and showed that once the MELD score exceeds 15, the survival benefit of transplant outweighs the risk, while

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Figure 2. **AASLD guidelines for risk stratification of patients with cirrhosis.**

Stages of chronic liver disease	No cirrhosis	Compensated cirrhosis		Decompensated cirrhosis	
		Lower risk of decompensation	Higher risk of decompensation	First decompensation	Further decompensation
Clinical features (ascites, VH or HE)	None	None	None	One event	>1 event or complication of event*
Histological diagnosis	F0–F2	F3/F4 (thin septa)	F4 (thin septa)	Clinical	Clinical
Hemodynamic features (HVPG mmHg)	3–5	5–10	>10 (CSPH)	>20 worse outcomes in VH	>20 worse outcomes in VH
Endoscopic features	None	No varices	± Varices	± Varices	± Varices

Risk of death

AASLD = American Association for the Study of Liver Diseases; CSPH = clinically significant portal hypertension; HE = hepatic encephalopathy; HVPG = hepatic venous pressure gradient; VH = variceal hemorrhage. (Reproduced from Kaplan DE, Ripoll C, Thiele M, et al, AASLD practice guidance on risk stratification and management of portal hypertension and varices in cirrhosis, *Hepatology*, volume 79, issue 5, pages 1180–1211 [http://doi.org/10.1097/HEP.0000000000000647], with permission.)

with MELD scores of less than 12 to 14, the 1-year mortality risk was significantly higher for patients who underwent transplant than those who did not (hazard ratio, 2.35; P < 0.001) (14).

The Child–Turcotte–Pugh (CTP) score can be a useful adjunct to MELD. The CTP score predated MELD and was used to determine the severity of cirrhosis for trial inclusion, risk stratification, and transplant prioritization before the advent of MELD in 2002. The score includes albumin, INR, bilirubin, ascites, and encephalopathy as categorical variables, resulting in a score from 5 to 15 that can be summarized as Child A (5 to 6), Child B (7 to 9 [cirrhosis]), or Child C (10 to 15). This classification continues to be used in practice, particularly for surgical risk assessment, although it is less commonly used than MELD (15). Because MELD can underestimate cirrhosis severity (for example, patients with hepatic hydrothorax can be "sicker than their MELD"), AST and AASLD recommend a CTP score of 7 for consideration of transplant referral (13). Moreover, the CTP score has been better validated for its associated longer-term (5-year vs. 90-day) mortality (11). Therefore, a mix of risk scores can be used to assess disease severity.

Presentation and Evaluation... Severity of cirrhosis can be estimated based on calculated scores, including MELD 3.0 or CTP score or class. Risk stratification is based on the presence of CSPH, which can be indicated by decompensation, direct measurement, or a combination of laboratory tests and elastography.

CLINICAL BOTTOM LINE

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Diagnosis and Management

What are common complications of cirrhosis to be managed in the inpatient setting?

The most common complications of cirrhosis include ascites and spontaneous bacterial peritonitis (SBP), variceal bleeding, portosystemic encephalopathy, acute kidney injury (AKI), and infection. Portopulmonary hypertension, hepatopulmonary syndrome, and other bleeding and clotting sequelae

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Table 1. **Criteria for Consideration of Referral for Liver Transplant***

Disease	*Criteria for Consideration*
Acute liver failure	Develops over a short period (<26 wk) Includes encephalopathy or coagulopathy (INR >1.5) King's College criteria and others can be used for risk stratification
Decompensated cirrhosis (e.g., ascites, encephalopathy, hepatopulmonary syndrome, hepatorenal syndrome)	MELD 3.0 score >15 or decompensation events; consideration for acute-on-chronic liver failure
Hepatocellular carcinoma	Cases should be reviewed by a multidisciplinary tumor board A single lesion measuring 2–5 cm or up to 3 lesions measuring <3 cm are "within Milan criteria" Downstaging criteria allow larger lesions to be treated before listing
Metabolic conditions with systemic manifestations (e.g., primary hyperoxaluria, familial amyloidosis)	Disease-specific criteria will be applied for these patients, who often receive MELD exception points
Acute alcohol-related hepatitis	Center-level variability in criteria, typically based on the original study

INR = international normalized ratio; MELD = Model for End-Stage Liver Disease.
** From reference 13.*

are also common. When patients present with decompensation in the inpatient setting, transplant evaluation should be considered, as decompensation portends worse prognosis. Although the threshold for transplant referral is center-dependent, typically any decompensation event or a MELD 3.0 score above 15 merits consideration.

What is the approach to assessment and management of variceal bleeding?

A diagnosis of CSPH in outpatients with cirrhosis should prompt consideration of NSBBs. Although expanded indications for NSBBs and updated guidelines for screening for varices have decreased bleeding rates, variceal bleeding remains a life-threatening emergency when it does occur (10, 16). AASLD, AGA, and other societies have developed guidelines to support the management of variceal bleeding (10, 17). These guidelines and others recommend that patients with suspected variceal bleeding warrant admission to the intensive care unit (ICU), large-bore intravenous (IV) access, and consideration for intubation if the airway is in jeopardy or risk for aspiration is high (18). A high index of suspicion should be used when a patient with cirrhosis presents with possible upper gastrointestinal (GI) bleeding, with the bleeding assumed to be variceal until proven otherwise. The patient should be started immediately on vasoactive therapy (octreotide in the United States; terlipressin where approved) and IV antibiotics to prevent infection (10). The transfusion goal should be a hemoglobin level of 7 g/dL (and not higher in the absence of shock) to avoid exacerbation of the portal hypertension that underlies the bleeding, unless there is a cardiac or other medical indication for a higher hemoglobin level (10). INR does not reflect coagulopathy for people with cirrhosis since the procoagulant and anticoagulant factors are both affected. Therefore, routine transfusion of fresh frozen plasma (FFP) is not recommended based on INR as it too can increase the portal pressure and exacerbate bleeding (19).

A retrospective study of 244 patients with variceal bleeding compared outcomes based on receipt of FFP transfusion. Multivariable regression models showed that FFP transfusion was associated with increased 42-day mortality (odds ratio [OR], 9.41 [95% CI, 3.71 to 23.90]), failure to control bleeding at 5 days (OR, 3.87 [CI, 1.28 to 11.70]), and odds of hospitalization exceeding

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1 week (adjusted OR, 1.88 [CI, 1.03 to 3.42]) (20).

AASLD and AGA recommend an upper endoscopy within 12 hours of presentation (10). In the absence of contraindications (such as QT prolongation), endoscopists often recommend preprocedure IV erythromycin to clear blood and clots from the gastric fundus to enhance visualization (10). Band ligation should be performed if there are high-risk endoscopic signs or in the absence of another credible source of bleeding (10). If bleeding cannot be controlled by ligation or injection of sclerosant, esophageal stenting or balloon tamponade should be performed, and the patient should be evaluated for emergent transvenous intrahepatic portosystemic shunt (TIPS) (10). Balloon tamponade was previously the mainstay of temporizing therapy, but more recently esophageal stent has been preferred (21).

A randomized controlled trial compared fully covered self-expandable metal stents with balloon tamponade for management of esophageal variceal bleeding that was refractory to endoscopic therapy in 28 patients with cirrhosis. The study found that stenting was significantly more likely to control bleeding (85% vs. 47%; $P = 0.037$*), with a nonsignificant reduction in adverse events in the stent group (15% vs. 47%;* $P = 0.077$*) (21). Subsequent meta-analysis has further supported this practice (22).*

Recent AASLD guidelines support consideration of TIPS in the context of variceal bleeding and offer detailed recommendations for its application in practice (23). However, the decision to offer TIPS is often best made in collaboration with an interventional radiologist, a transplant hepatologist, and a transplant surgeon since there are both risks and benefits. TIPS works by reducing portal hypertension by offering a conduit from the portal vein directly into the central vein. HVPG can be estimated using a catheter through the hepatic vein. When the measurement catheter balloon is inflated and occludes the hepatic vein, the "wedge pressure" reflects the sinusoidal pressure, which typically is a good estimate of the portal vein pressure. The free hepatic vein pressure, which is obtained by deflating the balloon in the hepatic vein, reflects the systemic venous pressure. The difference between the wedge and free pressures is the HVPG. A normal HVPG is 1 to 5 mm Hg, and 10 mm Hg indicates portal hypertension. The post-TIPS HVPG goal is typically 10 to 12 mm Hg, which reduces the risk for bleeding but is less likely to cause encephalopathy than a lower goal (23).

TIPS placement can be considered an emergency salvage procedure (when bleeding cannot be stopped), an "early" or "preemptive" procedure (within 24 to 72 hours of bleeding) to prevent subsequent bleeding, or a "rescue" procedure if the patient bleeds again within 5 days (10, 23). Although there are several notable contraindications to TIPS (**Table 2**), preemptive TIPS placement reduces mortality for patients with a CTP score above 7 and active bleeding on endoscopy or those with a CTP score of 10 to 13 (24, 25). Ongoing research seeks to clarify the timing of TIPS placement (for example, whether it can be extended to 5 days) (23). Patients who require TIPS placement and those with variceal bleeding should be considered for transplant evaluation.

Early TIPS placement is supported by a randomized controlled trial comparing it with traditional therapy for variceal bleeding. Among 63 patients with acute variceal bleeding at high risk for rebleeding after endoscopy, 32 received a covered stent within 72 hours after randomization and 31 received ongoing medical management and banding. The early TIPS group had reduced rebleeding (1 vs. 14 patients; $P = 0.001$*), reduced mortality (4 vs. 12 patients;* $P = 0.01$*), and increased 1-year survival (86% vs. 61%;* $P < 0.001$*) (24).*

AASLD guidelines for management of portal hypertension recommend that, after a patient is stabilized, clinicians

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should obtain cross-sectional imaging to evaluate thrombosis or cancer as potential contributors (10). In addition, after stabilization and control of the variceal bleeding, vasoactive therapy should be maintained for 2 to 5 days, with conversion to an NSBB before discharge or on day 5 (10). Carvedilol is the preferred NSBB based on recent comparative effectiveness data, with titration to a recommended dose of 6.25 mg twice daily or 12.5 mg once daily, although other NSBBs can be used if carvedilol is not tolerated (10). Antibiotics should be given by IV (generally ceftriaxone, but this should be in accordance with local resistance patterns) for 5 days or until discharge (10). Oral nutrition should be prioritized early, and proton-pump inhibitors (PPIs) should be discontinued in the absence of a strong indication (such as peptic ulcer disease) due to the increased risk for infection and encephalopathy with PPIs in cirrhosis (26). Although there is a potential role for prevention of encephalopathy using lactulose, perhaps especially in intubated patients, rapid removal of blood has not been recommended (27). Careful monitoring for early signs of encephalopathy is essential to enable timely and aggressive treatment.

Gastroesophageal varices (GOV) can occur in both the esophagus and the stomach. Type 1 GOV extend from the lesser curvature of the stomach and account for two thirds of gastric varices. Type 2 GOV extend from the greater curvature and are less common. There are also isolated gastric varices that do not connect to esophageal varices and ectopic varices that can arise elsewhere (such as the rectum or the duodenum). Gastric and ectopic varices may bleed at lower portal pressures and are typically not amenable to banding (with the exception of type 1 GOV lesions) and require vascular intervention (10). Because these lesions are at higher risk for rebleeding than esophageal varices, vascular intervention is generally considered (when appropriate), given the significantly reduced risk for rebleeding with TIPS compared with banding alone (10). Although medical management is similar to that for esophageal varices (vasoactive drugs, antibiotics), different approaches may be used endoscopically (such as cyanoacrylate) and different radiologic approaches are needed (TIPS with or without retrograde transvenous variceal obliteration or embolization) to treat such lesions. Likewise, when nonvariceal sources of bleeding are identified, such as peptic ulcer, portal hypertensive gastropathy, or gastric antral vascular ectasia, these should be treated endoscopically and antibiotic prophylaxis should continue, although the endoscopic therapies will differ, and PPIs may be indicated in these cases (10).

Per AASLD guidelines, after endoscopic band ligation, it is critical that patients undergo repeated endoscopies every 2 to 4 weeks until obliteration (10). Because 3 to 4 banding sessions are required to achieve obliteration, patients should leave the hospital with a follow-up appointment and plan. Likewise, TIPS requires follow-up with a hepatologist for ongoing assessment and evaluation.

What is the approach to diagnosis and management of ascites?

Ascites is the most common first decompensation event for people with cirrhosis and is graded as 1 (present only on imaging), 2 (clinically evident), or 3 (tense or large ascites) (28). Diagnostic paracentesis should be performed in people with cirrhosis and grade 2 to 3 ascites, regardless of the presence of symptoms or pain, because SBP is frequently asymptomatic and can precipitate other complications, such as encephalopathy or renal injury (28). Diagnostic paracentesis can be done without correcting INR or platelets since these parameters do not accurately reflect bleeding risk in people with cirrhosis (19). Cell count, differential, and cultures (inoculated in blood culture bottles) should be sent. The serum-ascites albumin gradient (SAAG) can be used to distinguish between ascites from the liver versus other causes (using a threshold of >1.1 g/dL to represent high SAAG ascites

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and protein to differentiate cardiac vs. hepatic source) (28).

The first, often overlooked step in managing volume overload and ascites in cirrhosis is restricting salt intake to 2 g per day (28). Patients often have hidden sources of salt in their diets (processed food, pickles, ketchup) that can precipitate admission. Dietary or nutrition services are an important component of these admissions. Similarly, inpatient teams need to ensure that the diet order is clear and the patient is receiving a truly low-salt diet in the hospital. In contrast to salt restriction, free water or fluid restriction is only indicated for the purpose of addressing hyponatremia, not for addressing ascites (28). Free water restriction is typically not recommended until the sodium level is below 125 mmol/L (28). A spot urine sodium–potassium ratio can help to distinguish diuretic refractoriness from dietary nonadherence; a ratio above 1 indicates a possible dietary component. It is important to note that sodium replacement is contraindicated in this setting as it exacerbates anasarca and acidosis and does not treat hyponatremia due to cirrhosis.

AASLD and AGA guidelines recommend giving albumin to everyone undergoing large-volume paracentesis (>5 L) at a dose of 6 to 8 g/L to prevent renal dysfunction and post-paracentesis circulatory dysfunction (26, 28). Albumin is also indicated for renal protection if SBP is diagnosed (discussed later). These guidelines are based on a large body of evidence outlined in a recent meta-analysis.

A meta-analysis identified 17 trials with 1225 total patients that compared albumin with alternative treatments for prevention of post-paracentesis circulatory dysfunction. Albumin reduced the incidence of circulatory dysfunction overall (OR, 0.39 [CI, 0.27 to 0.55]) and compared with each alternative treatment (for example, gelatin or saline). Both hyponatremia (OR, 0.58 [CI, 0.39 to 0.87]) and mortality (OR, 0.64 [CI, 0.41 to 0.98]) were also reduced in those receiving albumin (29).

In addition to dietary sodium restriction, diuretics are typically started after large-volume paracentesis (assuming there is no renal injury) (28). Spironolactone (or another aldosterone antagonist) is typically first-line treatment and can sometimes be used as monotherapy in a first episode of ascites. However, patients typically require the combination of spironolactone and furosemide (or another loop diuretic); guidelines recommend an initial spironolactone dose of 100 mg once daily, with a maximum of 400 mg/d (28). Spironolactone titration is not reflected in laboratory values and diuresis for 72 hours, so slower titration is required. The starting dose of furosemide is 40 mg once daily, with a maximum of 160 mg/d (28). The ratio of these diuretics is typically 100:40, but the doses and ratio may require adjustment based on potassium level (28). For example, patients with renal disease often require a higher furosemide–aldosterone ratio to maintain potassium homeostasis (28).

It is important to note that there is a risk for renal injury with diuresis; therefore, tenuous patients may require lower starting doses of 20 mg once daily for furosemide and 50 mg once daily for spironolactone (28). After the patient is at their goal weight and volume status, the diuretics should be titrated down to the minimal effective maintenance dose. Before discharge, patients require education about daily weight measurement, regular laboratory tests, and indications to call their clinician. For patients with refractory ascites, TIPS may be considered, although transplant referral is often recommended as well for anyone with decompensated cirrhosis. Contraindications to TIPS are outlined in **Table 2**, and the decision to proceed with TIPS placement should involve a transplant hepatologist (23).

Other contributors to ascites are important to consider. AASLD guidelines recommend performing cytology to detect

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Table 2. **Contraindications to TIPS***

Absolute
Congestive heart failure
Severe tricuspid regurgitation
Pulmonary hypertension (mean pulmonary pressure >45 mm Hg)
Polycystic liver disease
Active infection
Unrelieved biliary obstruction
Relative†
MELD 3.0 score >18
Hepatic encephalopathy
Hepatic tumor
Portal vein thrombus; obstruction of hepatic veins
Moderate pulmonary hypertension

MELD = Model for End-Stage Liver Disease; TIPS = transvenous intrahepatic portosystemic shunt.
** From reference 23.*
† Considered for the ascites indication but not acute bleeding.

malignant ascites and Doppler ultrasonography to evaluate portal flow and to rule out the presence of thrombus (28). In addition, medications such as nonsteroidal anti-inflammatory drugs (NSAIDs), amlodipine, angiotensin-converting enzyme inhibitors (ACEIs), angiotensin II–receptor blockers (ARBs), α1-adrenergic blockers, and dipyridamole can contribute to ascites or renal dysfunction and should be considered for discontinuation, particularly in the case of refractory ascites, because they can impair renal perfusion and promote diuretic resistance; nephrotoxins (for example, aminoglycosides) should also be avoided per these guidelines (28). Of note, ACEIs and ARBs need only be stopped in the context of refractory ascites (28). However, it is unclear when to discontinue carvedilol or other NSBBs in the context of refractory ascites. Although initial studies suggested increased mortality with NSBBs in the context of refractory ascites and there is agreement that they should be discontinued in the case of circulatory dysfunction, more recent data suggest they are often safe and beneficial to continue even in decompensated cirrhosis (30). However, due to the complexity of this decision, consultation with a specialist is likely warranted (28). Likewise, statin doses are generally reduced (for example, simvastatin, 20 mg/d) for people with indications and mild or medically controlled ascites (31). Due to the increased risk for rhabdomyolysis and myopathy, statins are generally stopped in the context of refractory ascites in patients with a bilirubin level above 3 mg/dL (32).

What is the approach to diagnosis and management of SBP?

SBP is diagnosed when a paracentesis sample includes at least 250 polymorphonuclear cells (PMNs) per microliter (28). All patients admitted with ascites should receive paracentesis, regardless of symptoms, and the fluid should be inoculated into culture bottles at the bedside and should be sent for cell count and differential (28). If SBP is diagnosed, antibiotics should be started for 5 days, regardless of whether the culture result is positive. AASLD recommends first-line antibiotics, including IV third-generation cephalosporins for inpatients (for example, ceftriaxone), for community-acquired SBP; for nosocomial SBP, broader-spectrum antibiotics, such as cefepime, meropenem, or piperacillin–tazobactam, are recommended (28).

Albumin infusions should follow a regimen of 1.5 g/kg of body weight on day 1 and 1 g/kg on day 3, which was found to be associated with decreased kidney injury and mortality (28). For patients meeting SBP criteria who have an organism in the initial cultures and are improving with antibiotics, paracentesis need not be repeated (28). Culture-negative neutrocytic ascites should be treated, meaning that even if the culture result is negative, SBP should be treated; AASLD guidelines recommend that paracentesis be repeated after 48 hours to ensure that the PMNs are decreasing by at least 25% and that the culture be repeated since treatment failure is common (28). Conversely, if the culture result is positive but the patient does not meet SBP criteria in terms of PMNs, this is called bacterascites. This should not be

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treated with antibiotics; rather, paracentesis should be repeated after 48 hours since bacterascites can progress to SBP in some cases (28). A "bloody tap" can be accounted for by subtracting PMNs from the count (for example, 1 PMN per 50 erythrocytes). Because SBP has been associated with PPI use, it is reasonable to consider stopping PPIs in patients without clear indications (33). AASLD and AGA recommend secondary prophylaxis after an episode of SBP, typically with daily norfloxacin (400 mg) or ciprofloxacin (500 mg), with double-strength trimethoprim–sulfamethoxazole falling out of favor due to emerging resistance (26, 28). This guidance is based on a randomized trial of norfloxacin versus placebo that showed a reduction in recurrent SBP (34). However, this study was done before the emergence of antibiotic resistance, leading to further studies of other agents and consideration of rifaximin as an alternative.

A 6-month randomized trial of rifaximin versus norfloxacin in 266 patients with ascites and SBP found significantly lower rates of recurrent SBP (4% vs. 14%; P = 0.04), mortality (14% vs. 24%; P = 0.044), and adverse effects (P = 0.033) in the rifaximin group (35).

Newer studies have found emerging antibiotic resistance and absence of improved outcomes, suggesting that secondary prophylaxis with absorbed antibiotics may carry more risks than benefits. For example, a study of 4673 veterans and 6708 patients in private health care with index SBP found that secondary prophylaxis was associated with significantly increased risk for non-SBP infections (36). Thus, ongoing studies are needed to evaluate secondary prophylaxis options, including rifaximin as a potential alternative to systemic antibiotics.

What is the approach to hepatic hydrothorax?

Similar to ascites, hydrothorax can develop and become infected in people with cirrhosis. Portal hypertension leads to fluid traversing fenestrations in the diaphragm and accumulating on either or both sides of the pleural space and may occur in the absence of ascites. Hydrothorax should prompt consideration of transplant referral since patients with this complication are often more ill than their MELD score indicates. Patients should have thoracentesis to ensure that the fluid is transudative and not infected (28). Spontaneous bacterial empyema is diagnosed with the same PMN count as SBP (≥250) and should be treated similarly (28). Management of noninfected pleural fluid includes sodium restriction and diuretics (28). Transplant and TIPS should be considered for hepatic hydrothorax that cannot be managed medically, but chest tubes, indwelling catheters, and pleurodesis are generally contraindicated due to risks for infection and further hepatic decompensation (28).

What is the approach to diagnosis and management of AKI and HRS?

Renal injury is a serious complication of portal hypertension. Prevention is thus key, and nephrotoxins (such as NSAIDs and aminoglycosides) should be assiduously avoided in this population (38). Likewise, diuresis should be done with caution and is often stopped if the creatinine level increases above 2 mg/dL. In the past, HRS was classified as type 1 or type 2, with rapid onset and higher mortality associated with type 1 HRS and slower, more chronic progression associated with type 2 HRS. Recent changes in nomenclature have shifted the conventions such that type 1 HRS is called HRS-AKI to offer clearer criteria (37). As with other AKIs, diuretics and nephrotoxins (such as NSAIDs) should be stopped, urine electrolytes and urinalysis should be sent, and a fluid trial should be given. Postrenal causes should also be assessed, particularly in patients with tense ascites.

If HRS-AKI is diagnosed (**Table 3**), terlipressin is now considered first-line treatment and should be considered, based on local approval criteria (38). When there are contraindications to terlipressin (such as heart failure, ischemic disease, hypoxia, or pulmonary edema) or it is unavailable, norepinephrine infusion should be considered (38). Because it is a potent vasoconstrictor, potential adverse effects of terlipressin include myocardial infarction and mesenteric ischemia. The patient should be reassessed for improvement on day 4, and the total course can be up to 14 days (38).

A randomized controlled trial compared 27 patients who received terlipressin with albumin and 22 patients who received midodrine and octreotide plus albumin for HRS. Recovery of renal function was 70% in the terlipressin group compared with 29% in the midodrine group (P = 0.01), leading to improved survival (39).

How are coagulopathy and thrombocytopenia managed?

AASLD and AGA offer guidelines for managing coagulopathy and thrombosis in the context of cirrhosis (19, 40). Although traditional measures of coagulopathy would lead clinicians to conclude that patients with cirrhosis have

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Table 3. **International Club of Ascites Diagnostic Criteria for HRS-AKI***

Cirrhosis, acute liver failure, acute-on-chronic liver failure
Increase in serum creatinine level of ≥0.3 mg/dL within 48 h or ≥50% from baseline value or urinary output ≤0.5 mL/kg of body weight for ≥6 h
No response to diuretic withdrawal and albumin volume expansion after 2 consecutive days (with albumin dosed at 1 g/kg of body weight per day)
Absence of shock
No recent nephrotoxic drugs
No evidence of structural renal disease (e.g., proteinuria >500 mg/d, microhematuria [>50 erythrocytes per high-power field], urinary injury biomarkers, or abnormal renal ultrasound)
Urine fractional excretion of sodium <0.2% (urine sodium may be inaccurate in people taking diuretics)

HRS-AKI = hepatorenal syndrome–acute kidney injury.
** Information from reference 37.*

increased bleeding risk, increasing evidence suggests that cirrhosis leads to a balance of procoagulant and anticoagulant deficiencies that often render patients more likely to develop thrombosis (for example, portal vein thrombosis). The fundamental concern for bleeding risk in this population has led to overtransfusion with platelets and FFP when this is often not required. Furthermore, overtransfusion risks volume overload and can exacerbate portal hypertension. Thus, these guidelines recommend against giving platelets or FFP for paracentesis (19). For low-risk procedures, including diagnostic endoscopy, a platelet count of 0.030 to 0.050 $\times$ 10^9 cells/L is typically considered sufficient, although the cut point varies among societies, with AGA recommending a count above 0.050 $\times$ 10^9 cells/L (19). The exceptions to this are high-risk surgeries or procedures or during active bleeding, when thromboelastography is used to indicate bleeding risk (19).

What nutritional support is needed for inpatients with cirrhosis?

AASLD guidelines specifically address nutrition and frailty (41). Sarcopenia (loss of muscle mass) and frailty are increasingly recognized as independent prognostic markers for people with cirrhosis. It is critical to assess nutritional status in this population and offer enteral nutrition when possible (including after acute management of GI bleeding). The guidelines recommend caloric intake of 25 to 35 kcal/kg and protein intake of 1.2 to 1.5 g/kg per day (41). Protein restriction is contraindicated in this population, and a late-night protein-containing snack is recommended to prevent ketosis (41). As with other populations, parenteral nutrition should be avoided when possible.

What is the approach to assessment and management of HE?

AASLD has a guidance document that addresses this topic in detail (42). In brief, HE is a decompensation event that can greatly impair quality of life for people with cirrhosis. Guidelines recommend dividing HE into minimal and overt subtypes, with the minimal subtype greatly underdiagnosed in this population (42). More specifically, HE can be graded using the West Haven scale, where grade 1 indicates minimal changes in level of consciousness or behavior (for example, day–night reversal) and can only be diagnosed by neuropsychiatric testing (for example, Stroop test). In contrast, grade 2 HE is clinically evident, with asterixis, drowsiness, and disorientation; grade 3 HE is characterized by incoherence but arousability; and people with grade 4 HE are comatose (42).

Encephalopathy can be diagnosed via clinical examination (for example, testing for asterixis) or neurocognitive testing (for example, a Stroop test) (42). Of note, asterixis is also associated with renal and pulmonary disease, so clinical context is important. Venous ammonia is not recommended to diagnose or follow treatment of HE (although a low ammonia level can help rule out HE in the right context) because it is often poorly correlated with arterial ammonia and is not sensitive or specific for HE (42). Other causes of hyperammonemia may include pulmonary or renal disease.

The presence of new encephalopathy should prompt a search for an underlying cause (for example, infection, metabolic, medication) so that any reversible cause can be addressed (43). Opioids and other psychoactive medications should be reconsidered, infection should be ruled out, and electrolytes should be corrected, although care must be taken to not correct hyponatremia too quickly (42, 43). Lactulose should be used as first-line therapy, targeting 3 soft bowel movements per day (42). Polyethylene glycol may be used as an adjuvant if patients are bloated and uncomfortable with high-dose lactulose or do not have the desired bowel movements with lactulose. Of note, diarrhea and dehydration can precipitate HE, so it is important to aim for 3 soft bowel movements per day rather than

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maximizing lactulose and causing diarrhea (42). Rifaximin has been shown to prevent rehospitalization for people admitted with HE as well as having other potential benefits, such as decreasing SBP risk (44). EASL and AASLD both recommend rifaximin, 550 mg twice daily, as an add-on to lactulose after an episode of overt encephalopathy (42).

Rifaximin was the subject of a Cochrane review that included 41 trials involving 4545 people with cirrhosis. Rifaximin and nonabsorbable disaccharide were superior to nonabsorbable disaccharide alone for all-cause mortality (risk ratio, 0.69 [CI, 0.55 to 0.86]; number needed to treat, 22), encephalopathy (risk ratio, 0.58 [CI, 0.48 to 0.71]), other serious adverse events, and length of hospital stay (45).

Although rifaximin is recommended, patients have had readmissions due to the discontinuity of medication availability. Therefore, in addition to prescribing rifaximin, it is important to ensure that patients have the resources in place to receive the medication (46). AASLD guidance recommends that HE should prompt consideration of discussing health care proxies for any future episodes that would render patients unable to make their own health care decisions (43). Depending on the degree of HE, it may be appropriate to discuss driving precautions or reassessment with patients and their families (42).

How is infection managed in inpatients with cirrhosis?

Cirrhosis is an immunocompromised state, so it is important to diagnose and treat infections early (26). As such, AASLD practice guidance documents, based on expert opinion, recommend a low threshold for infection work-up for patients hospitalized with cirrhosis, recommending diagnostic paracentesis, blood cultures, urinalysis, urine culture, and chest radiography in addition to work-up tailored to symptoms (for example, computed tomography for a suspected abdominal source) (18). Such a work-up should be repeated with further worsening or if there is not improvement in the expected time frame (18). Based on studies demonstrating risks of PPI use, this guidance recommends discontinuing these agents and minimizing Foley catheter use (18). Infections can be divided into typical community-acquired infections (such as pneumonia) and cirrhosis-related infections (such as SBP) (18, 28). Generally, the approach to antibiotic selection is similar to that for the general population, with a more aggressive approach to antibiotic therapy (18). Although there is increasing literature warning about the risks of antibiotic resistance and a growing awareness of the role of PPIs in increasing risk for SBP in patients with cirrhosis, as described earlier, there remains a need for aggressive empirical treatment of infections (18).

One third of 4238 inpatients with cirrhosis in a large global prospective study were found to have infections at admission, most commonly SBP (29%), pneumonia (17%), and urinary tract infections (14%). Less than half (41%) were culture-positive, and organisms were most commonly gram-negative (63%), with Escherichia coli, Klebsiella pneumoniae, *and* Enterococcus*; 40% of organisms were multidrug-resistant (47).*

How is pain managed in inpatients with cirrhosis?

Chronic pain is reported by up to 80% of people with cirrhosis and often contributes to reduced quality of life (43). Moreover, there are common misperceptions about how pain should be managed in the context of liver disease. For example, acetaminophen (up to 2 g/d) is generally first-line treatment for people with even decompensated cirrhosis, and NSAIDs should be avoided (43). Likewise, long-term opioids are not recommended for chronic pain, although they are acceptable for palliative or acute purposes (43). When opioids are used, care should be taken to preempt encephalopathy by using lactulose and preventing constipation. Other potential tools for analgesia include topical medications, behavioral approaches, and complementary medicine approaches like acupuncture. Newer guidance from AASLD offers detailed approaches to symptom management to assist with pain and other common cirrhosis symptoms (43). Consideration of palliative care services is recommended in all people with decompensated cirrhosis. Inpatient stays are often an opportunity to improve patients' access to such services and to ensure that patients are aware of their prognosis, have identified a surrogate decision maker, and have goals-of-care discussions documented (43).

What is the role of liver transplant evaluation during an inpatient stay?

Patients with decompensated cirrhosis should be considered for transplant and often warrant consultation with a transplant hepatologist for consideration of

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an inpatient evaluation if they are in the ICU or meet MELD criteria as set by local hepatology teams. Moreover, people with acute-on-chronic decompensation should be considered more urgently. The exact criteria for liver transplant evaluation and listing are often center-specific and complication-dependent. It is important to communicate with the hepatology team and to understand when to transfer patients to a transplant center for further evaluation. For example, anyone with acute-on-chronic liver disease, fulminant liver failure, or a high MELD score who has not been deemed to be ineligible should be considered for transfer (18).

What is the role of palliative care in patients with decompensated cirrhosis?

AASLD recently released its first guidance document about palliative care in cirrhosis, which recommended that palliative care consultation be offered to all patients with decompensated cirrhosis (43). AGA also has made recommendations about the importance of palliative care (48). These recommendations are based on data suggesting that palliative care is associated with improved quality of life, reduced cost of care, better support for caregivers and patients, and reduced readmissions (43). AASLD guidance also notes that transplant evaluation and concurrent listing do not preclude palliative care (43), based on studies demonstrating benefits for patients receiving palliative care consultation concurrently with transplant listing.

What are considerations at discharge of inpatients with cirrhosis?

Safe discharge requires an individualized plan. People with cirrhosis often have unplanned readmission within 30 days due to inadequate planning, early discharge, lack of continued care, or medication errors (49). It is important to ensure that patients and caregivers are educated about cirrhosis and self-care. Pharmacy-led medication reconciliation (50) can be helpful for providing the needed education, removing all hepatotoxins, and avoiding herbal and over-the-counter supplements and medications. Likewise, nutrition guidance (for example, a low-sodium diet) can prevent readmission, and inpatient settings are opportunities to offer alcohol cessation services. It is important to ensure close follow-up with the hepatology team and consider case management and social services (51). Social determinants of health often dictate the extent to which a patient can follow a specified plan. For example, healthier, low-sodium foods may be more expensive, and transportation to appointments can be a barrier.

Diagnosis and Management... Cirrhosis often remains undiagnosed until complications of decompensation occur, so recognizing and staging liver disease is an important component of hospital medicine. Transplant should be considered for all patients with cirrhosis complications, such as hepatic hydrothorax, ascites, and encephalopathy, with more urgent evaluation for acute-on-chronic liver failure and HRS-AKI. Variceal bleeding is an often-fatal complication of cirrhosis and therefore requires a high index of suspicion, ICU care, antibiotics, vasoactive medications, transfusion to a hemoglobin level of 7 g/dL (unless a higher level is warranted), and urgent endoscopy within 12 hours of presentation. TIPS can be considered for refractory ascites or to help with management of variceal bleeding, but there are key contraindications; early echocardiography can help determine TIPS candidacy. Any inpatient with ascites requires diagnostic paracentesis to rule out SBP. Ascites requires sodium restriction and diuretic management; fluid restriction is typically reserved for people with a sodium level below 125 mmol/L. Because of the balance of procoagulant and anticoagulant factors in cirrhosis, transfusion of FFP and platelets should be reserved for high-risk procedures (not paracentesis) and should ideally be based on thromboelastography rather than laboratory values alone. Terlipressin is the current standard of care for HRS-AKI. Medication review can help to determine underlying contributors to decompensation, such as opioids or NSAIDs. Encephalopathy in inpatients requires efforts to ensure continuation of rifaximin for discharge. Palliative care services should be considered for all patients with decompensated cirrhosis.

CLINICAL BOTTOM LINE

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Practice Improvement

What do professional organizations recommend with regard to prevention, diagnosis, and management of cirrhosis in inpatients?

Guidelines from American and European liver disease societies offer specific recommendations as well as evidence levels for the guidance described here. As referenced earlier, AASLD has guidelines and guidance documents that focus on noninvasive assessment of liver disease severity (6, 7), ascites management (28), encephalopathy (42), acute-on-chronic liver failure (18), symptom management and palliative care (43), and portal hypertension and varices management (10), among other topics.

What quality measures are used in cirrhosis?

The AASLD Practice Metrics Committee recently used a modified Delphi process to select 41 measures from among those identified through a literature review, including 9 for referral, 20 for evaluation and waitlisting, 7 for waitlist management, and 5 for organ acceptance (52). In addition, quality measures focusing on management of cirrhosis outside the transplant space have been recommended by the AASLD Practice Metrics Committee, based on a similar process (53). These recommendations highlight management of portal hypertension, hepatocellular carcinoma surveillance every 6 months, hepatitis B vaccination, and frailty assessment and include outcomes such as readmission and mortality. The U.S. Department of Veterans Affairs has also developed measures, including screening for and treatment of portal hypertension, hepatocellular carcinoma surveillance, and 30-day readmission (54).

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In the Clinic
Tool Kit

Inpatient Management of Patients With Cirrhosis

Patient Information

https://medlineplus.gov/cirrhosis.html
Information on cirrhosis from the National Institutes of Health's MedlinePlus.

https://patient.gastro.org/cirrhosis
Overview of cirrhosis from the American Gastroenterological Association.

https://www.mayoclinic.org/diseases-conditions/cirrhosis/symptoms-causes/syc-20351487
Patient information on cirrhosis from the Mayo Clinic.

https://www.niddk.nih.gov/health-information/liver-disease/cirrhosis
https://www.niddk.nih.gov/health-information/informacion-de-la-salud/enfermedades-higado/cirrosis
Information on cirrhosis in English and Spanish from the National Institute of Diabetes and Digestive and Kidney Diseases.

Information for Health Professionals

https://journals.lww.com/hep/fulltext/2025/01000/aasld_practice_guideline_on_blood_based.29.aspx
American Association for the Study of Liver Diseases practice guideline on blood-based noninvasive liver disease assessment of hepatic fibrosis and steatosis.

https://journals.lww.com/hep/fulltext/2025/02000/aasld_practice_guideline_on_imaging_based.30.aspx
American Association for the Study of Liver Diseases practice guideline on imaging-based noninvasive liver disease assessment of hepatic fibrosis and steatosis.

https://journals.lww.com/hep/fulltext/2024/05000/aasld_practice_guidance_on_risk_stratification_and.22.aspx
American Association for the Study of Liver Diseases practice guideline on risk stratification and management of portal hypertension and varices in cirrhosis.

https://journals.lww.com/hep/fulltext/2024/06000/aasld_practice_guidance_on_acute_on_chronic_liver.25.aspx
American Association for the Study of Liver Diseases practice guideline on acute-on-chronic liver failure and the management of critically ill patients with cirrhosis.

https://journals.lww.com/hep/fulltext/2021/09000/malnutrition,_frailty,_and_sarcopenia_in_patients.38.aspx
American Association for the Study of Liver Diseases 2021 practice guidance on malnutrition, frailty, and sarcopenia in patients with cirrhosis.

https://journals.lww.com/hep/fulltext/2021/08000/diagnosis,_evaluation,_and_management_of_ascites,.38.aspx
American Association for the Study of Liver Diseases 2021 practice guidance on diagnosis, evaluation, and management of ascites, spontaneous bacterial peritonitis, and hepatorenal syndrome.

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WHAT YOU SHOULD KNOW ABOUT CIRRHOSIS

What Is Cirrhosis?

Cirrhosis is a condition where the liver becomes permanently scarred, usually after years of damage from things like alcohol use, hepatitis, or fatty liver disease.

How Is the Presence of Cirrhosis Assessed?

Doctors assess cirrhosis by combining several types of information:

- Medical history and physical examination, looking for signs such as jaundice, fluid retention, or mental confusion
- Blood tests, which can show how well the liver is working
- Imaging tests of the liver, such as ultrasound, CT scan, or MRI, which can suggest scarring
- Elastography, which measures liver stiffness as a sign of scarring
- Liver biopsy, although this is rarely necessary nowadays

What Complications Are Commonly Seen in Hospitalized Patients With Cirrhosis?

In hospitalized patients with cirrhosis, common serious complications due to impaired liver function may include:

- Fluid buildup in the stomach (called ascites) or swelling in the legs
- Bleeding, especially from enlarged veins in the esophagus or stomach
- Infections due to weakening of the immune system
- Confusion or sleepiness due to toxin buildup
- Kidney problems

How Are Cirrhosis-Related Complications in Hospitalized Patients Diagnosed?

Cirrhosis-related complications are diagnosed using a combination of symptoms, physical examination findings, laboratory tests, imaging, and sometimes special procedures.

- Fluid in the abdomen (ascites): Found by physical examination or ultrasound; sometimes fluid is sampled with a needle to check for infection

- Bleeding from enlarged veins (varices): Diagnosed with an upper endoscopy (camera examination of the esophagus and stomach)
- Confusion (hepatic encephalopathy): Diagnosed based on mental status examination and blood tests
- Infections: Detected through blood tests, cultures, imaging, and testing of abdominal fluid if ascites is present
- Kidney problems: Diagnosed with blood and urine tests and by identification of kidney toxins

How Are Cirrhosis-Related Complications in Hospitalized Patients Treated?

Cirrhosis-related complications in hospitalized patients are managed with multiple measures.

- Fluid buildup in the stomach (ascites): Low-salt diet, water pills, and sometimes draining of fluid with a procedure
- Bleeding from varices: Medications to lower pressure in the veins and endoscopy to directly treat bleeding veins
- Confusion (hepatic encephalopathy): Laxatives and antibiotics to reduce toxins along with treatment of underlying triggers, such as infection
- Infections: Antibiotics
- Kidney problems: Optimization of fluid volume in blood vessels and avoidance of triggers (toxins and medications)

Questions for My Doctor

- What is causing my cirrhosis?
- What medications should I take or avoid?
- What changes to my diet should I make (salt, protein, fluids)?
- What can I do to slow worsening of cirrhosis?
- Am I a candidate for a liver transplant evaluation?

For More Information

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MedlinePlus
https://medlineplus.gov/cirrhosis.html

American Gastroenterological Association
https://patient.gastro.org/cirrhosis

Mayo Clinic
https://www.mayoclinic.org/diseases-conditions/cirrhosis/symptoms-causes/syc-20351487

National Institute of Diabetes and Digestive and Kidney Diseases
https://www.niddk.nih.gov/health-information/liver-disease/cirrhosis
https://www.niddk.nih.gov/health-information/informacion-de-la-salud/enfermedades-higado/cirrosis

Patient Information

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